Brozek and Keys (1957) calculated average weights for years 1 and 2 (before test subjects stopped smoking) and for years 3 and 4 (after test subjects stopped smoking). A weight change of +1.18 kg was reported for the control group and of +5.65 kg for the group who quit smoking cigarettes. The weight change for subjects who quit smoking showed the effect of body weight on smoking cessation.

Kershbaum (1966) reported that there was a greater increase in blood free fatty acid (FFA) concentration with cigarette than with cigar smoking. Inhaling caused a greater FFA response than no inhaling with both cigars and cigarettes. With tobacco containing glucose labelled with C-14, there was a greater absorption of C-14 with cigarette smoking. The greater output was attributable to differences in smoke inhalation and nicotine absorption.

Aziz et al. (1978) found that resting urinary level of total catecholamines was slightly higher in a smoker group than in a nonsmoker group. Smoking 20 cigarettes increased urinary catecholamine output in all subjects. The increase was largest in hypertensive smokers, in normoreactive smokers and nonsmokers. Smoking 2 cigarettes increased the number of hyperreactors among hypertensives by 60% and the number of hyperreactors among normotensives by about 20%.

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levels; these results confirm the association between smoking and elevated levels of serum IgE. Ferson et al. (1979) reported that low natural killer cell activity and immunoglobulin levels in smokers may be related. Miller et al. (1982) showed that cigarette smoking causes reversible changes in immunoregulatory T cells. Corberand et al. (1979) investigated the effect of smoking on the functions of polymorphonuclear leukocytes in 68 subjects including smokers and nonsmokers. Phagocytosis, oxygen consumption, and bactericidal activity were normal in smokers. Myeloperoxidase and neutrophil alkaline phosphatase activities were not changed. Nitroblue tetrazolium reduction and serum lysozyme levels were slightly increased in smokers. However, the capillary tube random migration was depressed and this was further aggravated by intensive smoking. Tobacco smoke may act directly on one or more unidentified target sites of polymorphonuclear leukocytes, which probably plays a role in the development of bronchopulmonary disease in heavy smokers. Rasp and Clawson (1978) studied the reversibility of the adherence of alveolar macrophages from smokers in 33 smokers and nonsmokers using a standardized nylon fiber assay and scanning electron microscope. The adherence of alveolar macrophages from cigarette smokers was uniformly decreased. However, after 2 months abstinence the defect was not found, which was not attributable to factors in lavage fluids and was not apparent in polymorphonuclear leukocytes. The surface of alveolar macro-

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smokers showed a deficit in surface active material. The volume of surfactant rapidly returned to the same levels as those of nonsmokers when the subject stopped smoking. There was no qualitative difference in the lipid analysis of surfactant from smokers and nonsmokers. However, the total lipid content, especially lecithin, was seven times less in smokers than nonsmokers.

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Hart et al. (1976) reported that the mean antipyrine half-life was lower in smokers than in nonsmokers. Cigarette smoking significantly enhanced antipyrine disappearance rate; smoking contributed to the large variation in rates of drug metabolism observed in these subjects.

Smith and Landau (1978) concluded that carbon monoxide exposure from cigar or cigarette smoke was a frequent cause of an increased erythrocyte volume and/or a decreased plasma volume detected in smokers.

Warren et al. (1982) studied the effect of smoking on the immune system. The relationship between smoking and serum IgE levels was investigated. Skin tests, total serum IgE levels, and a respiratory questionnaire were used with 1,768 subjects, including smokers and nonsmokers. For subjects who had no skin test reactions or history of asthma or hayfever, the geometric mean total IgE levels were 14.8 U/ml for men and 11.9 U/ml for women. IgE levels among nonsmokers, former smokers, and current smokers varied significantly when skin reactivity and smoking history were considered. The percentage of subjects with elevated total IgE was significantly higher in smokers than in nonsmokers regardless of skin reactivity. Although the IgE levels were significantly higher in smokers than nonsmokers, there was no relationship between intensity of smoking and IgE

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D. Withdrawal Effects

1. Physiological Changes
2. Mood
3. Psychomotor Performance
4. Cortical Arousal
5. Eating Behavior and Weight Control

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antidiuresis. Smoking 2 cigarettes increased secretion of antidiuretic hormone (Burn and Grewal, 1951). Nicotine was found to be a stimulus for both vasopressin and neurophysin secretion in some normal individuals, and the release mechanisms for these two peptides may be closely linked (Husan et al., 1975).

In 34 of 60 habitual smokers, in vitro thrombus formation time was found to be decreased following smoking; in 23 subjects the time was unchanged; and in 3 subjects the time was increased (Engleberg, 1965). However, when blood was repeatedly drawn over a period of several hours, there was variability of thrombosis time. The disparity seen between thrombus formation times and clotting times after smoking emphasized that thrombis and coagulation are different processes. Increased thrombotic tendency may be mediated via the nicotine-induced secretion of epinephrine. This hyperthrombotic state may be a major etiological factor in the increased incidence of acute myocardial infarction in smokers.

Tanzen and Nilssen (1975) reported that after 12 hours abstention from tobacco, smokers had the same fibrinolytic activity as nonsmokers. Of 71 heavy smokers, 31 refrained from smoking during 8-9 weeks. Neither in those who abstained nor in controls did the fibrinolytic activity differ from that initially recorded. The smoking of six cigarettes was associated with increased fibrinolytic in superficial hand veins.

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Rowe et al. (1980) reported that intravenous administration of nicotine resulted in no effect on plasma vasopressin, a transient increase in mean blood pressure and heart rate associated with symptoms of light-headedness. Low and high nicotine cigarettes increased plasma vasopressin (high nicotine cigarettes had a greater increase). Symptoms of light headedness after high nicotine cigarettes and nausea after low nicotine cigarettes were reported. All 3 intravenous doses of nicotine yielded transiently increased blood pressure and heart rate of similar magnitudes. Smoking both cigarettes was followed by a transient increase in blood pressure. The increase above basal plasma vasopressin is greater after high nicotine cigarettes than low nicotine cigarettes. These results confirm that smoking in the absence of nausea, vomiting or decreases in blood pressure induces vasopressin secretion. This effect may be mediated by an airway-specific mechanism.

Burn et al. (1945) reported that six out of seven subjects experienced inhibition of diuresis for two to three hours after smoking one, two or three cigarettes, dependent on individual sensitivity. An experiment with two subjects showed the same effect after intravenous injection of nicotine in the amount equal to the amount that produced the effect after smoking. Walker (1948) also showed that 1 or 2 cigarettes produced

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Wilkinson and Johnson (1971) reported that in control pentagastrin infusions in which 9 normal subjects and 5 duodenal ulcer patients simulated smoking, gastric acid secretion, heart rate, and blood pressure all remained constant. Cigarette smoking produced inhibition of pentagastrin-stimulated gastric acid secretion in normal subjects and in patients with duodenal or peptic ulcers. In 11 normal subjects, acid secretion decreased by a mean of 38%; in 10 patients with duodenal ulcers, by a mean of 21%; in 4 patients with gastric ulcers, by a mean of 18%. Acid output decreased after smoking in 25 subjects. Inhibition produced by 2 cigarettes was not greater than inhibition produced by 1 cigarette. Inhibition was as great in smokers as in nonsmokers. No significant change in acid secretion was observed during constant pentagastrin infusion in normal subjects and in patients with peptic ulcers who puffed on non-nicotine cigarettes. Acid concentration decreased by a mean of 12% in 11 normal subjects; by 14% in 10 duodenal ulcer patients; and by 17% in 4 gastric ulcer patients. Mean pepsin secretion decreased by 31.0% in 11 normal subjects and by 44.9% in 10 duodenal ulcer patients. Smoking 1 cigarette elicited decreased acid secretion of 2%, 78% and 42% in subjects who received nicotine acid tartrate (1, 3, and 4 mg). Acid secretion decreases after infusion of nicotine (3 mg) similarly to the decrease produced by cigarette smoking.

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phages from smokers showed marked alterations that could affect the ability to adhere. Alveolar macrophages from smokers had a defect in structure and adherence that may influence their function and may account for the increased yield of alveolar macrophage from cigarette smokers.

To evaluate the role of oxidants from alveolar macrophages on the development of emphysema, alterations in the oxidative metabolism of alveolar macrophages were studied in 51 nonsmokers and 32 smokers (Hoidal et al., 1981). The superoxide anion produced in smokers was greater than that from nonsmokers. This also occurred before and after stimulation by bacteria or phorbol myristate acetate. However, oxygen uptake and glucose oxidation by unstimulated and stimulated alveolar macrophages from smokers were the same as that from nonsmokers. Because intracellular superoxide dismutase was increased in alveolar macrophages from smokers, the increase of superoxide anion did probably not result from lack of anion scavenging agent in the cells. The lysis of fibroblasts by alveolar macrophages from nonsmokers was completely prevented by addition of intracellular superoxide dismutase and catalase, confirming the importance of superoxide anion release. The elastase and lysozyme activities in alveolar macrophages were studied in 27 smokers and nonsmokers (Hinman et al., 1980). Macrophages, particularly those in smokers, were found to synthesize a calcium-dependent activity against synthetic

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D. WITHDRAWAL EFFECTS

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2. GASTROINTESTINAL

Schnedorf and Ivy (1938) studied the effects of nicotine on gastrointestinal activities in smoking and nonsmoking normal human subjects, patients with peptic ulcer, and dogs. Results indicated that smoking, but not subcutaneously injected nicotine (0.4 mg), stimulates salivation in most smoking and nonsmoking subjects. Hunger contractions of the stomach in human subjects tended to decrease for 15-60 minutes following smoking one cigarette. While subcutaneous nicotine injection (1 mg) did not inhibit such contractions in dogs, inhalation of cigarette smoke inhibited the gastric motility promptly. Inhibition was blocked by sectioning of the vagi above the diaphragm in dogs, indicating reflex control of these contractions via vagal motor pathways. In normal smokers, there was no significant effect of smoking on emptying time. However, gastric acidity was significantly decreased. Neither gastric emptying time nor acidity were significantly affected by smoking in the ulcer group. Subcutaneous injection of 1 mg nicotine decreased acid secretion in one of three dogs. Subcutaneous nicotine injection (0.2, 0.4 or 1 mg) or smoking two cigarettes in dogs had no effect or decreased acid secretion during fasting. Smoking one to two cigarettes and subcutaneous or intravenous injections of 0.2 to 1 mg of nicotine had no effect on bile or pancreatic secretion in dogs, except when marked changes in blood pressure were produced by nicotine.

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E. Summary and Effects on Soldier Readiness

1. Soldier Readiness
2. Summary
3. Recommendations

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acid output as well as an increase in volume and in chloride output. A study of 4 subjects showed that smoking stimulated both the parietal-cell and non-parietal-cell components of gastric secretion.

Murthy et al. (1977) reported that smoking resulted in a transient increase in basal acid secretion, but was significant only for ulcer subjects. This increase was followed by a nonsignificant decrease for both groups. Fluid and bicarbonate secretion were inhibited during the smoking period, but returned to control levels within 30-60 minutes in the ulcer groups and within 60-90 minutes in the nonulcer group. There was no difference between the two groups in the degree of pancreatic secretion inhibition during smoking. Basal pancreatic secretion inhibition correlated with plasma nicotine concentrations. Plasma levels of secretion and gastrin were not altered by smoking. Debas et al. (1971) found that the mean plateau output of acid was 4.56 millimoles/15 minutes or 42% of the maximum response. Stimulation of acid secretion occurred following the smoking hour. Pepsin response reached 180% of the control value. It seems unlikely that smoking may exert a harmful effect on the normal human stomach, but that conclusion may not apply to the ulcer patient. This was confirmed by Rack and Sonnenberg (1983) who found that during fasting, gastric juice, and, during infusion of pentagastrin, the gastric concentration and output of

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initial increase in these symptoms during the first day of abstinence. Significant increases in symptoms were reported in the evening for the placebo group in comparison to morning and afternoon responses and in contrast to the nicotine gum group. Nicotine gum appeared to have ameliorated withdrawal symptoms compared to a continuing increase in symptoms in placebo subjects.

Shiffman and Jarvik (1980) studied participants in a smoking cessation clinic during the first two weeks of abstinence. Results showed a strong diurnal variation in craving for cigarettes, with craving lowest early in the morning and rising to a peak around 7:00 PM. Many symptoms appeared within two hours of withdrawal. Severity of all symptoms decreased the first week, followed by leveling off or increase in the second week. Two groups, totally abstinent smokers and partially abstinent smokers who decreased their cigarette consumption by an average of 60%, reported similar levels of symptom severity. The totally abstinent group experienced a notable decrease in symptoms during the first week; whereas, the partially abstinent group maintained withdrawal symptoms.

A preliminary study of three subjects suggested that the tobacco withdrawal syndrome is reliable and can be studied using within-subject designs (Hughes et al., 1984). The majority of

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E. SUMMARY AND EFFECTS ON SOLDIER READINESS

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bound N-acetylneuraminic acid (NANA) were similar in smokers and in nonsmokers. Acute nicotine administered intravenously or by smoking had no effect on gastric NANA. This study suggests that neither chronic nor acute consumption of nicotine may affect gastric turnover or adherence of mucus to the mucosa. Whitecross et al. (1974) studied the effect of cigarette smoking on gastric secretion in 13 control subjects and 8 subjects with chronic peptic ulcers. Gastric juices were collected from each subject after smoking 4 cigarettes in 1 hour. The output of acid, pepsin and mucus was not significantly different in either group or between the two, and was not influenced by the tar and nicotine content of the cigarettes. In the control subjects biliary reflux was more marked in the hour that the subject smoked. Consequently, smoking does not seem to influence gastric secretions or ulcer healing. Sonnenberg and Husmert (1982) investigated the effects of nicotine on gastric mucosal blood flow and acid secretion in 13 males. Doses were administered via intravenous nicotine infusion and cigarette smoking, with blood flow measured by gastric neutral red clearances. Nicotine reduced volume secretion, acid secretion and neutral red clearance in a dose-dependent manner. Smoking five cigarettes elicited an effect similar to an intravenous dose of 5 ug/kg/hr of nicotine. Results seem to show that nicotine increases blood supply to the gastric mucosa relative to the reduced gastric secretion. Nicotine may exert its ulcerogenic action via other mechanisms than change in acid secretion and gastric mucosal blood flow.

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irritable, restless, dizzy, and hungry after 2 hrs of abstinence from cigarettes. They also reported they were less social and less able to concentrate. The 2 mg gum helped to alleviate some of these effects of depression, irritability, and problems in socializing. The 0.5 mg gum group exhibited a decrease in heart rate and excreted epinephrine; the 2 mg gum produced a smaller decrease in heart rate, but did not ameliorate the decrease in epinephrine excretion. The onset of hunger and absence of detectable increases in emotional irritability are consistent with the results of previous studies on the effects of nicotine chewing gum in decreasing withdrawal symptoms. Although the gum alleviates irritability and depression it does not seem to prevent a short term increase in hunger. A small dose of nicotine may prevent an increase in irritability but a higher intake may be required to prevent an increase in hunger.

Schneider and Jarvik (1984) studied the time course of smoking withdrawal symptoms in relation to nicotine replacement by nicotine chewing gum and compared subjects receiving either nicotine or placebo gum. Signs and symptoms of withdrawal were measured on a scale consisting of the following items: irritability, hostility, anxiety, depression, frustration, restlessness, annoyance, disorientation, insomnia, mental concentration, alienation, fluctuations in mood, concern about weight, and craving for cigarettes. All subjects reported an

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pressure and weight were measured every two weeks. Questionnaires were given to detect changes in physical activity, diet, and anxiety. The experimental group showed an increased anxiety level. Both groups had significantly lower norepinephrine levels after the study than at the beginning.

2. Mood

Smoking withdrawal phenomena can often be detected within hours of the last cigarette. As little as 90 minutes of abstinence may increase emotional irritability (Schecter and Rand, 1974). Hutchinson and Emley (1973) measured jaw clenching in smokers attending a withdrawal clinic at weekly intervals after subjects stopped smoking, and reported an increase in the frequency of spontaneous jaw contractions after smoking cessation, which was correlated with verbal reports of irritability. Switching to an ultra-low nicotine cigarette may induce the subjective and physiological effects of nicotine withdrawal. In a study of these effects over a 10-day interval, West et al. (1984a) found that the plasma nicotine concentration decreased by 60% after switching. Switching to a low-nicotine cigarette was accompanied by a significant increase in hunger, a decrease in mean heart rate of slightly more than 6 beats per minute, and a significant increase in skin temperature of 1.8 degrees C. Mean epinephrine concentration tended to be decreased (not statistically significant), but other common cigarette

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Heimstra et al. (1977) examined the effects of smoking on driving performance by comparing ad lib smokers, abstaining smokers, and nonsmokers in a 6 hour simulated driving test. On a variety of measures of tracking and vigilence, there were no significant differences between the performance of nonabstaining smokers and nonsmokers, but abstaining smokers exhibited decreased tracking and vigilance performance. Myrsten et al. (1977) found that verbal learning was essentially unaffected over five days of smoking abstinence.

4. Cortical Arousal

Herning et al. (1983) studied effects of acute tobacco withdrawal of 10-17 hours on EEG changes. Cigarettes were smoked at 30 minute intervals either freely (Group 1) or at the rate of one puff per minute (Group 2). The time course of EEG spectral changes after smoking a single cigarette was followed by a one day experimental session. The effects of repeated nicotine cigarette smoking on EEG and the duration of nicotine effects after switching to placebo cigarettes were tested in a two-day session. Theta and alpha power increased significantly following tobacco abstinence and were decreased by nicotine cigarette smoking but not by placebo smoking. The peak alpha frequency was decreased more after abstention than after smoking a nicotine cigarette. Decreases were not always significant. The increased theta power during abstention may correlate with drowsiness and a

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In both smokers and nonsmokers, smoking increased colon motility. Following smoking, significant increases in gastric retention (emptying rate), gastric acidity, untoward cardiovascular effects (fainting after smoking on an empty stomach) and increase in colon motility were observed.

Ivey and Triggs (1978) reported that nicotine was well absorbed in 15 minutes during intragastric administration at pH 9.8. Absorption was accompanied by untoward effects of nausea and vomiting, and delay in gastric emptying. Rapid cigarette smoking and i.v. nicotine decreased gastric acid secretion. Neither oral administration, i.v. infusion (4 mg/hr of nicotine base), nor smoking 3-5 cigarettes per hour significantly altered the gastric mucosal barrier (as measured by gastric ionic fluxes and potential differences). Grines and Goddard (1978) reported that smoking a cigarette increased the rate at which liquid left the stomach, suggesting that the rate of acidification of the duodenum after a meal might be correspondingly more rapid. Solid did not accompany the liquid and did not buffer the excess leaving the stomach. Pathogenesis of duodenal ulcer and delay in healing may be associated with cigarette smoking. Piper and Raine (1959) studied the maximum secretory response to histamine estimated during the 2nd hour after smoking 4-6 filter cigarettes. Secretion in the 2nd (smoking) hour showed an increase over the basal hour of over 100% in free acid and total

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for the smoking condition, subjects smoked one cigarette during the 10 minutes immediately prior to the first half hour period and one cigarette during each 10 minute break thereafter. During the nonsmoking condition, subjects smoked one cigarette prior to the first performance task but did not smoke again for the four hour session. Visual tracking was the only task component showing a statistically significant decrease in the smoking withdrawal condition, although there was a tendency for performance in monitoring red lights to decrease during withdrawal over the first three half hour periods; vigilance performance in monitoring red lights tended to increase with time in the smoking condition. In tracking performance, the adverse effect of smoking withdrawal was independent of a trend toward decreased performance with time, which occurred in both smoking and nonsmoking conditions. Performance in monitoring red lights and arithmetic tasks increased with time in the smoking condition. Subjective ratings of attentiveness were significantly higher when subjects smoked. Arousal was not significantly affected by either smoking or time of testing but smoking tended to increase arcusal and withdrawal to decrease arousal over the course of the session. Levels of tiredness, tension, boredom, and irritation were higher at the ends of the sessions than before but were unaffected by smoking withdrawal. The higher heart rates and higher attentiveness scores obtained when subjects smoked suggest that decreased arousal in the absence of smoking may be a mechanism for detrimental effects of smoking withdrawal in habitual smokers.

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decreased heart rate, and decreased arterial blood pressure accompany withdrawal. Relaxation and feelings of tranquility have been associated with smoking, while increased aggression has been found during withdrawal. Relaxation by smoking has also been associated with decreased skeletal muscle tension at rest, but increased resting muscle tension has also been detected. Effects of nicotinic stimulation and withdrawal on decision-making and manual dexterity have been addressed in experiments measuring simple choice reaction times, response time, and arithmetic and letter cancellation tasks. Smoking decreased inhibition associated with key tapping, an effect that may have implications for reaction time testing. Both smoking and nicotine withdrawal decrease hand steadiness, a motor effect that may decrease both reaction/response times and manual dexterity. Increased errors have been reported by arithmetic and letter cancellation tests under both conditions of smoking and withdrawal.

Learning and recall reflect the complex interaction of information acquisition, storage, and retrieval. Both smoking and withdrawal have produced mixed results. Under some conditions, learning and recall is enhanced and under other conditions, they are impaired. State dependent learning occurs wherein information learned under the effects of nicotine is recalled better while smoking than while abstaining.

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Sogani and Joshi (1965) studied whole blood clotting time, recalcified plasma clotting time, prothrombin time, platelet adhesiveness, fibrinogen content, and fibrinolytic activity. Changes before and after tobacco consumpton were seen in all tests performed, but only the effect on platelet adhesiveness and fibrinolytic activity were significant. An increased tendency to coagulation and decreased fibrinolytic activity were evident in all the tests performed and with three modes of tobacco consumption.

Levine (1976) reported that in 11 smokers, no platelet aggregation responses were measured 10 and 20 minutes before and after smoking a lettuce-leaf cigarette. Before and after smoking a standard cigarette compared to the change in aggregation after lettuce-leaf cigarette smoking, the increases in aggregation were maximal at 10 and 20 minutes and at or near baseline by 30 minutes. None of the 11 subjects developed an increase in blood FFA after the lettuce-leaf cigarette. After the standard cigarette, all 11 subjects had increased FFA. The time of maximal FFA increase was 30 minutes after smoking. In three subjects, the increase in FFA occurred only after the increase in platelet aggregation was noted. The data suggest a possible direct casual link between cigarette smoking and arterial thrombotic disease.

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withdrawal symptoms such as irritability, depression, and inability to concentrate, were not observed. The major subjective effect of lower nicotine concentration was increased hunger.

Several studies have shown that nicotine deprivation plays a major role in the cigarette withdrawal syndrome and that relatively low levels of nicotine replacement aid in amelioration. In a study of the effect of nicotine gum on withdrawal symptoms, West et al. (1984b) found that following a 24-hour abstinence from cigarettes, with nicotine supplied as 0.5 or 2 mg in chewing gum, the low nicotine gum group was significantly different in measures of depression, irritability, and sociability than the high nicotine group. There were no significant differences in measures of restlessness, dizziness, ability to concentrate, and hunger. The 2 mg gum group had significantly less increase in irritability and depression and significantly less decrease in sociability than the 0.5 mg gum group. The 2 mg gum group also exhibited a significant increase in hunger and decrease in ability to concentrate, compared to the changes in the 0.5 mg group. The average decrease in heart rate was 14.4 beats per minute in the 0.5 mg gum group but only 9.7 beats per minute in the 2 mg group. There was a slight but nonsignificant increase in skin temperature in the 0.5 mg gum group. Subjects in the 0.5 mg gum group rated themselves as having become increasingly

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the signs and symptoms of tobacco withdrawal occurred during abstinence, returned to baseline when smoking resumed, and then recurred during a second abstinence period. Variables such as setting, expectancy, and method of cessation may have influenced the syndrome, but these variables were held relatively constant. In comparing baseline and abstinence conditions, there were consistent decreases in heart rate and consistent increases in insomnia, caloric intake, irritability, restlessness, and drowsiness. The 4 signs and symptoms that did not occur consistently were orthostatic hypotension, craving, and difficulty concentrating in one subject each and anxiety in two subjects. Two total discomfort measures indicated that intensity of withdrawal discomfort was similar across trials.

3. Psychomotor Performance

Several studies of complex performance have shown significant adverse effects of smoking withdrawal on visual tracking; there is additional evidence that short-term withdrawal from smoking can produce decrements in attention and vigilance, choice reaction time, and selective attention (Mertens et al., 1983). Mertens et al. (1983) studied the effects of smoking withdrawal during a four hour interval on the performance of healthy smokers in flight related tasks at a simulated cabin altitude of 6,500 ft. Each experimental session consisted of six 30 minute tasks separated by 10 minute intermissions. During the test session

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E. SUMMARY AND EFFECTS ON SOLDIER READINESS

1. Soldier Readiness

The concept of soldier readiness is defined as the ability to perform any specialized and/or routine military duty. There is a wide range of tasks that may be required to be performed in preparation for and during combat. Therefore, the effects of nicotinic stimulation and withdrawal on combat readiness and performance review the major findings of the present report on (a) central nervous and psychological; (b) cardiopulmonary and musculoskeletal; and (c) metabolic and gastrointestinal functions.

2. Summary

a. Central Nervous and Psychological Functions

Smoking nicotine produces a transient stimulation followed by depression of both sympathetic and central nervous structures. Circulating epinephrine and vasopressin secreted from the adrenal medulla and neurohypophysis augment sympathetic and central nervous stimulation. Increased blood glucose level that may be secondary to epinephrine secretion could contribute to the mood elevation, although epinephrine may also act directly on the central nervous system to produce positive affect. Negative affect is associated with nicotine withdrawal. Although the etiology is complex and has not been systematically investigated, there is evidence that decreased blood epinephrine and excretion,

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APPENDIX A: CODING INSTRUCTIONS

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Systematic measurements of performance that directly relate to combat and soldier readiness have generally not been performed. Therfore, future experiments should involve careful analysis of tasks necessary to soldier readiness and studies designed to simulate appropriate conditions. In selection of tasks to be studied, it should be emphasized that physiological systems are inherently efficient; there is usually considerably less opportunity for improvement than for impairment of performance. Detection of improvement may be difficult unless situations are designed to be somewhat stressful in order to impair performance under control conditions. Therefore, the ideal task designed to study performance may be one that is capable of showing both improvements and impairments in performance. Use of frequent control measurements and training intervals prior to testing may decrease the possibility of training effects confounding performance data derived from smoking subjects.

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D. WITHDRAWAL EFFECTS

Cessation of smoking produces a set of signs and symptoms that have been termed the tobacco withdrawal syndrome. An increasing number of investigations have linked many of these objective and subjective effects of decreases in accustomed nicotine intake, or nicotine withdrawal. The onset of the syndrome appears to be rapid, with changes in mood (Schecter and Rand, 1974) and performance (Myrsten et al., 1972) evident as early as two hours after withdrawal. A wide variety of unpleasant side effects are reported by habitual smokers who stop smoking, including craving for tobacco, irritability, restlessness, dullness, sleep disturbances, gastrointestinal disturbances, anxiety, and impaired concentration, judgment, and psychomotor performance. The onset of symptoms may occur within hours or days of quitting and may persist for a few days to several months. Additional objective signs include decreases in heart rate and blood pressure, increased rapid eye movement (REM) sleep, and decreased frequency of EEG activity.

This section focuses primarily on the acute effects of nicotine withdrawal, although some long-term effects are cited when relevant.

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APPENDIX B: STATISTICAL RATING SCALE

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1. Physiological Changes

A range of physiological changes occurs upon nicotine withdrawal. Physiological signs of smoking cessation that have been reported consistently in several studies include decreased heart rate, increased skin temperature as a result of peripheral vasodilatation, increased predominance of EEG slow-wave activity, decreased excretion of epinephrine, and decreased blood glucose concentration 30 minutes after an oral glucose load as late as one month after cessation (West, 1984; Myrsten et al., 1977). Abstaining smokers or those who switched from high-nicotine to low-nicotine brands exhibited decreased heart rate and blood pressure. A decrease of around 60 percent in blood nicotine levels was found to be associated with relatively mild withdrawal effects (West, 1984). Elgerot (1978) reported that abstinence was accompanied by a decreased arousal, indicated by decreases in both epinephrine and norepinephrine excretion, with these decreases paralleled by increased emotional irritability. Puddey et al. (1984) reported on 66 smokers divided into two groups to study the effect of smoking on blood pressure. The experimental group was asked to stop smoking on entry into a six-week withdrawal program. The control group continued their normal smoking habit during this time before entering a similar program. Laboratory assessment at baseline and at the end of the study included a cold pressor test and assays for epinephrine, norepinephrine, cortisol, and prolactin. During the study blood

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discomfort and decreased pulmonary function under some conditions, and filtering of tobacco smoke has been used to effectively decrease pulmonary impairment. Recently, central respiratory drive has been shown to be decreased by smoking. These effects probably limit tolerance for physical exercise in synergism with cardiovascular factors.

c. Metabolic and Gastrointestinal Functions

Smoking may increase the fasting blood glucose level in normal and especially in diabetic subjects. Increased circulating catecholamines, growth hormone, cortisol, and prolactin concentration may both mediate and exacerbate this hyperglycemic diabetogenic effect. These hormones may increase the blood free fatty acid concentration and produce adipokinesis in smokers. Mobilization of fat from body adipose tissue depots may decrease the fat body mass and contribute to the lower total body weight of groups of smokers versus groups of nonsmokers, although this has not been systematically studied. However, decreased threshold for bitter and increased threshold for sweet tastes associated with smoking may contribute to decreased caloric intake and control of body weight. Nicotine withdrawal seems to increase body weight, supporting the theory that an important reason for smoking is weight control in some individuals.

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4. Routine simple statistical procedures including descriptions of control and experimental samples, analysis of variance, t-test, probit analysis, regression analysis, sample distributions, and P-values for statistical significance have been described and used.

5. Exceptionally sophisticated statistical methods including multivariate analysis, curvilinear plots, nonparametric tests, or complex statistical procedures have been used.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Similarly, there was considerable variability in experimental conditions; diurnal changes associated with peak cigarette consumption at a particular time of day, diet and eating behavior, compliance of subjects with protocol, dose of nicotine, and amount of smoke inhalation were generally not controlled. Testing was often perfo med after smoking intervals when the blood nicotine level had peaked. Therefore, performance may have been evaluated when the blood nicotine level might have decreased by as much as 50% within 10 minutes. From a pharmacological viewpoint, future studies should be performed before, during, immediately after, and even further after the last puff of the cigarette concomitantly with blood nicotine analyses. It may take 3 hours for the blood nicotine level to decrease to zero following smoking; therfore, less than 3 hours abstinence may compound results by the effects of residual nicotine. Conversely, studies performed after 3 hours abstinence may be interpreted as data derived from smokers during nicotine withdrawal.

Smoking and nicotine effects on learning and memory have been especially difficult to interpret because of the complex process of learning that has usually not been addressed in study protocols. Memory is usually inferred from the persistence of the effects of experience until the time of a recall test. Memory impairment has four stages: (1) information input; (2)

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

APPENDIX C: COMPUTERIZED DATABASE USER INSTRUCTIONS

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

one month after smoking cessation. Six of the subjects gained weight during the month after smoking cessation; mean weight increased from 83.29 kg to 87.33 kg. The subject whose weight remained constant markedly increased his activity levels. Significant decreases in heart rate, 30 minute postprandial blood glucose level, protein bound iodine, oxygen consumption, and serum calcium were observed. These results indicate that the metabolic changes associated with cessation of smoking involve decreased basal oxygen consumption. With no change in caloric intake or physical activity, these metabolic changes would produce weight gain. A study of 300 normal, middle-aged men showed a significant 9.3 lb (4.23 kg) gain over a 5 year interval in those who voluntarily stopped smoking in comparison with a control group who did not stop smoking (Brozek and Keys, 1957). Smoking tended to decrease the perceived need for food. This interpretation was based on experimental observations of the inhibition of gastric hunger contractions on smoking and the increased tobacco consumption by individuals on low-calorie diets. A longitudinal analysis of 57,032 women confirms the weight changes found in longitudinal studies of men and indicates the weight gain associated with smoking cessation is permanent (Blitzer et al., 1977). Light smokers, who smoke one-half pack a day, may expect to gain about 5 pounds if they stop; moderate smokers may expect to gain 15 pounds. Heavy smokers, consuming two or more packs per day, gained about 30 pounds. Cross-sectional analysis showed that former smokers were 3.2 to 3.8 pounds heavier than current smokers.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

1) Adding new records to the database.

2) Modifying records in the database (either in your own personal collection of them or in the entire database).

3) Generating polished output formats and tables.

To obtain further information about these features, see your database administrator to request Datatrieve documentation and training materials.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

information storage and consolidation; (3) information maintenance and retention; and (4) information retrieval and recall. Using preacquisition doses of nicotine, it may not necessarily be concluded that nicotine had modified learning unless a companion study rules out changes in sensory processing, motivation, and motor output. A partial solution may involve administration of nicotine or cigarettes prior to acquisition, but to test recall after nicotine has been excreted would not affect motor/sensory performance at the time of recall. Postacquisition doses of nicotine may not affect information input, but rather information retrieval and storage.

Future studies of cigarette smoking should be designed to study the effects of carbon monoxide independently of nicotine on the same subjects. Carbon monoxide is a colorless and odorless gas produced by the incomplete combustion of organic matter. The smoke from one cigarette can contain up to 21.4 mg of carbon monoxide. Cigarettes have concentrations ranging from 0.1-2.5 mg nicotine. In the database described herein, there are few examples of studies so designed, but when performed, parallelism and synergism between the effects of carbon monoxide and nicotine have been demonstrated.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

- That the Performance Database tape be copied into your directory. This transfers raw data to you. (You can do this yourself if you call the computer center to obtain the tape number and if you know the commands for mounting and dismounting tapes.)

- That the computer center modify your "LOGIN.COM" file to permit you to use Datatrieve. (You can amend your LOGIN.COM yourself by adding the following two lines:

  ```
  $DTR32="SYS$SYSTEM:DTR32.EXE"
  $ASSIGN/PROCESS "CDD$TOP.DTR$USERS.
  [Your Username]"CDD$DEFAULT
  ```

  Note that your own VAX Username must be used for the underscored text (not brackets) above.

- That the Computer Center copy the Performance Database record formats into the VAX Common Data Dictionary. Specify whether you want all users to have access, or only yourself. This permits you to use (and modify) the data copied earlier.

If you wish to access the Database from a computer nearer your terminal than Frederick, MD, you can:

- Download the above files to disk or tape on your local VAX, or

- Request the Frederick Computer Center to copy the files to tape and ship the tape to you.

Please observe that the Performance Database is proprietary software of USAMRDC. No copying of, downloading to, or sharing of access to the database is authorized without written USMRDC content.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

3. Recommendations

The database described herein is composed of human research on the effects of smoking and nicotine on soldier performance and readiness for combat. Some studies were designed to determine whether cigarette smoking or nicotine affected performance; others were designed to determine what aspect of smoking affected performance. Thus, the majority of studies were not based upon any particular theoretical framework, and the database reflects an accumulation of fragmentary evidence acquired from unrelated studies using different, nonstandardized methodologies. Such a structure makes it difficult to make meaningful comparisons between studies because of the large data gaps identified. Some of these gaps may be filled by animal studies; there is a large body of information from experimental animals in the literature. Experimental animal data has been excluded from the present database by design, and its inclusion would contribute significantly to the analysis of the effects of smoking and nicotine on human performance.

In the human studies described herein, most experimental populations were heterogenous with respect to age, sex and anthropometric variables. Such designs contributed to the great inter-individual variations reported. Multivariate statistical analysis was performed infrequently, but when performed, confirmed that future studies should use homogeneous subject populations to decrease variability.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

STAT:	Enter strings of 2-letter codes indicating statistical parameters (each code separated by space) from the list of statistical codes).
STATRAT:	Enter a number in the range 1-5 indicating the complexity of statistical analysis (Appendix B).
DOCTYPE:	Enter a single letter (J, B, or R) indicating whether the source is a book, journal, or report.
COND:	Enter a single letter (e.g., G, F, or P) indicating whether the condition (readability) of the ACI copy of the hit document is good, fair, or poor.
CAT:	Enter a combination of a letter followed by a period and a string of numbers each separated by a period (e.g., C.1.3.5) not to exceed 12 characters including periods from the list of category codes.
PERFN:	Enter the name of each performance variable not to exceed 48 characters each (limit 10 performance variables).
SUBJNO:	Enter the number 0-999 indicating the total number of subjects.
MALENO:	Enter a number 0-999 indicating total number of male subjects.
FEMALENO:	Enter a number 0-999 indicating total number of female subjects.
AGE:	Enter a number 0-4 indicating the age range of subjects.
SUBJTYPE:	Enter a 1-letter code indicating whether the subjects were smokers (S), nonsmokers (N), light smokers (L), medium smokers (M), heavy smokers (H), combination of L, H, or M = X, combination of S and N = Y.
ASPER:	Enter the pre-experimental abstinence interval in hours as numbers only (limit 4 digits).
DISCAT:	Enter the disease category (not to exceed 10 characters) abscribed to subjects that are not normal healthy subjects; if normal and healthy enter "NORMAL".

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

"	Number of Females	FNO
"	Age of Subjects	AGE
"	Subject Type	SUBJTYPE
"	Abstinence Period (from Nicotine)	ABSPER
"	Disease Category	DISCAT
"	Species	SPECIES
"	Drugs (includes all subfields)	DRUGS
"	Drug Name	DRUG
"	Dose	DOSE
"	Route	ROUTE
"	Form of Drug	DFORM
"	Level of Exposure	LEVEL
Index	Number of Index Terms	ITNO
"	Index Terms (0-50 Terms)	ITERM
Annote	Abstract (0-100 lines)	ABSTRACT

All fields in the Performance Database are searchable.

III. GAINING ACCESS TO THE PERFORMANCE DATABASE

The database is stored on one volume of 160-bpi magnetic tape at the USAMRDC Computer Center in Frederick, Maryland. To access the Frederick data you must first have authorized access to that VAX. Then you must call the computer center to request three things:

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

substrate which was distinct from polymorphonuclear leukocyte elastase. This activity was enhanced in smokers. Smokers' macrophages contained serine-proteinase activity that had an inhibitor profile similar to that of polymorphonuclear leukocyte elastase. The macrophages from smokers secreted 5 times more lysozyme and contained more lactate dehydrogenase activity than those of nonsmokers. These data show that polymorphonuclear elastase can become associated with the alveolar macrophage, which could assume a protective role against unrestrained elastinolysis. Martin (1973) studied alveolar macrophages from 11 smokers and 13 nonsmokers for altered morphology and increased acid hydrolase content. Crystalloid, refractile cytoplasmic inclusions of autofluorescent material were found in fewer than 5% of macrophages from nonsmokers while 30 to 95% from smokers contained this material. Multinucleated giant cells were found in the lavage of 3 smokers. The macrophages from smokers had acid hydrolase concentrations that were increased as much as 6 times normal. These increases correlated with the amount of daily cigarette consumption. The increased acid hydrolase concentrations in the cells from smokers indicate a mechanism by which chronic pulmonary disease may be related to smoking.

Finley and Ladman studied pulmonary surfactant layers taken from the lungs of smokers and nonsmokers by making an analysis of lipids. When compared with nonsmokers, endobronchial lavage of

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

mild version of stimulant withdrawal syndrome. Changes in alpha activity have been attributed to characteristic differences in arousal or cognitive processing of smokers; changes in alpha power and peak frequency may be simply secondary to the theta power changes. The dominant alpha frequency in abstaining smokers was decreased compared to the smoking of a nicotine cigarette, but decreases were not always statistically significant.

Soldatos et al. (1980) monitored the sleep patterns of five male smokers for five days of abstinence and found that sleep latency and time awake after sleep onset decreased. The improvement in sleep efficiency may have resulted from decreased catecholamine concentrations after withdrawal from smoking.

5. Eating Behavior and Weight Control

Population studies have shown that smokers tend to weigh less than nonsmokers (Blitzer et al., 1977). Changes in eating behavior and weight gain following cessation of cigarette smoking are two of the most widely studied effects of nicotine withdrawal. Weight gain is well documented, but whether it results from increased appetite and food intake or metabolic changes, or a combination of both, has not been precisely determined. Glauser et al. (1970) studied metabolic changes associated with smoking cessation in seven men at the time of and

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Attentiveness and rapid information processing have been studied in combination with visual and auditory stimuli, requiring motor responses of subjects. Some components of attention and rapid information processing have been associated with increased speed and accuracy, while others have produced performance decrements. During monotonous tasks, vigilance has been increased and the normal decremental performance observed over a prolonged time course has been ameliorated by nicotine and smoking. There is evidence that after three hours, the blood nicotine level is decreased virtually to zero, and testing over prolonged time may, therefore, provide information on performance during withdrawal.

Effects of nicotine and smoking that increase CNS arousal and withdrawal effects that decrease CNS arousal may also affect abilities to remain alert during monotonous, prolonged tasks and to learn new tasks. It has been shown that interindividual variations in performance during smoking may be related to smoking under high arousal versus low arousal conditions. Indeed, personality differences among smokers and measures of extroversion and neuroticism have been correlated with regulation of CNS arousal by nicotine and smoking.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Search #2 Find all records with the author Ashton. Print the bibliographic citation and abstract.

```
DTR> FIND PERFORMANCE WITH AUTHOR GE "ASHTON" AND
CONT> AUTHOR LT "B"

[11 records found]

DTR> PRINT ACC OF CURRENT

DTR> PRINT ANNOTE WITH ACC=34 OR ACC=59
```

Note that text parameters you input (e.g., Performance="reaction time" are enclosed in quotes. Numeric parameters (e.g., ACC=324) do not need quotes.

Note too that operators LT, LE, GT, GE can be used with either alpha or numeric fields, a capability that permits searching of non-indexed records.

Search #3 Find all records with author Ashton where the performance variable was reaction time. Print the bibliographic citation and abstact.

```
DTR> FIND PERFORMANCE WITH PERF_VAR="REACTION TIME"
CONT> AND (AUTHOR GE "ASHTON" AND AUTHOR LT "B")
```

(Note: "CONT" is how the system prompts you for a continuation line on an incomplete command.)

The basic syntax for searching, thus, is:

COMMAND } DATABASE "WITH" } FIELDNAME } OPERATOR } PARAMETER(S)

or

FIND ... [Subdatabase] ... WITH ... [Fieldname] ... AND/OR/GT/GE/LT

LE ... [Parameter]

You may print any search this way.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Line #1 --

Enter the author's last name followed by first initial followed by middle initial for all authors; separate each author by comma (e.g., Vogel J A, Minner D F, Speight Y).

Line #2 --

Enter the title of "hit" document capitaliizing the first letter of the first word only; followed by period (The effects of nicotine on human performance.).

Line #3 --

Enter the full name of source, followed by volume, followed by issue number in parentheses ( ), followed by colon (:), followed by page range, followed by comma, followed by publication year, followed by period (e.g., Proceedings of the Society for Experimental Biology and Medicine, 88(3):888-898, 1978).

When no issue number given (e.g., 88:888-898, 1978).

Line #4 --

Leave blank.

Line #5 --

Enter 70 character text (not to exceed 100).

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Smoking and nicotine effects on attentiveness and alertness have produced mixed effects on learning tasks. Smoking subjects have demonstrated selectivity of memory processing to information that is perceived to be irrelevant more than relevant and to information presented in the peripheral more than the central visual fields.

Decreased visual acuity from smoking has been associated with decreased size of visual fields, widening of retinal angioscotomata, decreased light sensitivity of both rods and cones, and speeding of dark adaptation. In contrast, rapid visual information processing may be enhanced by smoking. The frequency at which flickering light forms a single image (flicker fusion frequency) is increased in some and decreased in other smokers, and decreases in flicker fusion frequency have been correlated with other visual defects. Flicker fusion frequency may increase with CNS arousal and both increases and decreases have been reported with passive smoking. Movement detection, temporal judgments and estimation of velocity and distance of moving targets have been impaired under some conditions and improved under other conditions. When performing complex visual and auditory monitoring tasks simultaneously, only the auditory task was impaired by nicotine and smoking.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

APPENDIX A: CODING INSTRUCTIONS

Performance Record

ACC:	Enter number 1-999 stamped on document.
LOC:	Leave blank.
AU:	Enter last name followed by space followed by first initial followed by space followed by middle initial, without punctuation (each AU not to exceed 60 characters, up to a limit of 6 AU).
TI:	Enter title of document on 3 lines (each line not to exceed 60 characters). Numbers are acceptable.
SO:	Enter journal name, or book title without punctuation in combinations of numbers and letters (not to exceed 70 characters); source of report may be name of government agency or DTIC TECHNICAL REPORT. Abbreviations are not acceptable.
VOL:	Enter volume number of combination of numbers and letters not to exceed 8 characters to indicate source collection series; leave blank if source is a book or report without volume indicated.
SPAGE:	Enter the first page number of the hit document (not to exceed 4 digits).
LPAGE:	Enter the last page number of the hit document (not to exceed 4 digits).
PUBYR:	Enter the year of publication (not to exceed 4 digits).
ISSUE:	Enter the issue of the source as either a number or combination of numbers and letters indicating a date (not to exceed 15 characters, e.g., 15 MAR 1978).
PUBLISHER:	Enter the name of the publishing company or government agency if source is either a book or a report, in combinations of letters and numbers if necessary without punctuation (not to exceed 48 characters); if source is journal, leave blank.
LANG:	Enter "ENG".

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

IV. LOGGING ON AND OFF THE PERFORMANCE DATABASE

To access the Database:

- Log on the proper VAX.
- At the operating system ("$") prompt, type "DTR32"

```
$DTR32
```

(Underscored text indicates your input.)

The system will open Datatrieve as follows:

```
VAX Datatrieve V3.0
DEC Query $ Report System
Type HELP for help.

DTR>
```

The "DTR>" prompt means that you are ready to use Datatrieve. To exit Datatrieve, type "Exit."

```
DTR> Exit
```

This will return you to the operating system. To abort any commands or activities you wish Datatrieve to interrupt, type CTRL-Y.

To obtain help, simply type "Help Error" after an error message. The system will give you an analysis of what you did wrong. You can also obtain help messages by typing "Help." The system will give you a menu of help messages to choose from.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

SPECIES: Enter "HUMAN".

DRNO: Enter the number 1-10 indicating the number of drugs studied; ifmore than one dose or form of drug, enter each dose and/or form as a separate drug.

DRUG: For each drug number enter under the field DRNO, enter the scientific names of the drugs (generic) as specifically as possible in combinations of letters and numbers if necessary (not to exceed 36 characters); name the organic moiety first (e.g., nicotine); all records will have at least one drug as some form of NICOTINE (and possibly CARBON MONOXIDE).

Index Record

ACC: Enter number 1-999 stamped on "hit" document.

ITNO: Enter number 0-50, indicating the number of index terms assigned to hit document (limit 50 index terms).

ITERM: Enter each descriptive index term as combination of letters and numbers if necessary (not to exceed 55 characters); the number of ITERM entries must be exactly equal to the number indicated by ITNO entry.

Annotation Record

ACC: Enter number 1-999 stamped on "hit" document.

ABSNO: Enter the number 1-100 indicating the number of 70 character lines required to print the abstract (annotation).

ABSTRACT: Enter the annotation giving the bibliographic citation first, then skipping a line and entering the abstract as a single paragraph.

Enter the bibliographic citation in the following format:

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

V. ACCESSING THE DATABASE

To read and search records, you must first "ready" the database as follows:

```
DTR> Ready Performance
DTR> Ready Index
DTR> Ready Annote
```

This matches up each data file with the formatting codes to permit searching and output.

VI. SEARCHING THE DATABASE

Below are some sample searches to illustrate the use of the Performance Database:

Search #1 Find all records where the Performance Variable (PERF_VAR) studied was "reaction time." Print the bibliographic citation and abstract

```
DTR> FIND PERFORMANCE WITH PERF_VAR="REACTION TIME"

[4 records found]

DTR> PRINT ACC OF CURRENT

DTR> PRINT ANNOTE WITH ACC=324 OR ACC=533 OR
CONT> ACC=546 OR ACC=585
```

Note: "Current" designates the set created by your most recent "FIND" command.)

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

whose fields can be merged and mingled freely. This arrangement permits the database to add new fields at a later date, and also gives the Performance Record certain options and special features it would not otherwise have.

The fields in the database are:

Subdatabase	Field Contents	Field Name (for Searching)
Performance	Accession	ACC
"	Author (1-n)	AUTHOR
"	Title	TITLE
"	Source Title	SO
"	Start Page (0-n)	SPAGE
"	Last Page (1-n)	LPAGE
"	Publication Year	PUBYR
"	Issue Number	ISSUE
"	Publisher	PUBL
"	Language	LANG
"	Statistical Rating	STAT
"	Document Type	DOCTYPE
"	Document Condition	COND
"	Category Code	CAT
"	Performance Variables (1-n)	PERF_VAR
"	Number of Subjects	SUBJNO
"	Number of Males	MNO

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

APPENDIX B

STATISTICAL RATING SCALE

The computerized database includes a quantifying statistical rating scale (STATRAT field) assigned by the principal investigator, that is indicative of the quantitation of results described in each source document. Values range from 1 to 5 evaluated by the following criteria:

1. Quantitative description of data is lacking.

2. Basic statistical methods and parameters are lacking, but the data have been described quantitatively by measurement average values, distribution, sample size, and number of controls. Scatistical analysis of variance has neither been described nor given.

3. Detailed description of statistical method is lacking, but statistical analysis appears to have been performed. Standard error of the mean, chi square (with degrees of freedom), correlation coefficients, or P-values for levels of statistical significance have been appended to results.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Search #4 Print the entire database.

You may do this with Annote or Index with the command form:

```
DTR> PRINT ANNOTE WITH ACC GE 0
```

You may not do this with the subdatabase Performance because it requires a special report format for proper display of data. See your database administrator to request this. Should you request a "dump" of the Performance subdatabase, you may see the data with uncontrolled wrapping.

(Use CTRL-Y if you inadvertently request this or any other unwanted and lengthy search.)

For reasons that are clear, it is always better to search the database first before printing your data. At times, doing this may require that you skim or scan a set before printing. This is described in Search #5.

Search #5 Print only part of a search set, so you can skim its contents to see if these are the records you want.

```
DTR> FIND PERFORMANCE WITH PERF_VAR
CONT> "REACTION TIME"

[14 records found]

DTR> SELECT 1
DTR> PRINT AUTHOR
DTR> SELECT 2
DTR> PRINT AUTHOR
```


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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Note that skimming simple fields such as ACC can be done with PRINT ACC OF CURRENT. Subfields such as Author require that you select records one at a time.

Search #6 Print the authors and all performance variables for accession #4.

1) DTR> FIND PERFORMANCE WITH ACC=4
   DTR> PRINT ACC,AUTHOR,PERF_VAR

or

2) DTR> PRINT ACC, AUTHOR, PERF_VAR
   CONT> OF PERFORMANCE WITH ACC=4

Note that to print more than one filed at a time, you must link the fieldnames with commas, and place them with the word "of" before the subdatabase you are searching. In addition, you must request the ACC as your first field to display.

When requesting multiple fields in the performance subdatabase, Datatrieve will automatically format your output in columnar fashion.

If you request fields that cannot fit the 80-character line-length on your CRT, you will receive an error message or inappropriate wrapping. To correct this, request an output format from your database administrator.

VII. FURTHER ACTIVITIES WITH THE PERFORMANCE DATABASE

The Performance Database is a powerful and flexible application of Datatrieve. It is best to practice and experiment with it to get a real feel for the many ways to incorporate it into your work.

There are a variety of other functions Datatrieve permits that are not covered here:

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

I. INTRODUCTION

The Performance Database is a collection of on-line bibliographic citations and abstracts housed on the USAMRDC Computer Center's VAX 11/780 at Frederick, Maryland. The Database covers all published literature regarding the effects of selected chemical agents on human performance.

At present, the database deals with the agent nicotine, and contains 453 records representing the most current and authoritative literature and non-public literature (e.g., DTIC) concerning the effects of nicotine. The literature search from which these 453 items were selected covered over 1,100 pieces of the most up-to-date information on the subject, including journal articles, private research notes, and research reports.

The literature search was conducted during the period October 1985-January 1986, so the database may be considered to reflect the state-of-the-science through mid-1986. (At that time, update searching would be needed to confirm the currency of the nicotine data.)

II. THE PERFORMANCE DATABASE RECORD

The Performance Database uses the VAX DATATRIEVE Database Management System (TM). Technically, the database consists of three linked subdatabases -- Performance, Index, and Annote --

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000

Anxiety, age, and anthropometric classification may be important determinants of inter-individual variations in body weight changes associated with withdrawal from smoking. Increased blood vasopressin (antidiuretic hormone, ADH) levels associated with smoking may act synergistically with catecholamines contributing to systemic hypertension. This may be especially significant for increased peripheral vascular resistance because of the cutaneous vasoconstriction associated with smoking. The antidiuretic action of vasopressin concentrates urinary solutes, increases specific gravity, and may contribute to increased circulatory blood volume and hypertension.

Increased fibrinolysis and platelet aggregation produces a hyperthrombotic state that may contribute to the increased incidence of myocardial infarctions and other arterial thrombotic diseases associated with smoking.

Decreased immunological responsiveness associated with smoking may increase susceptibility to both acute infections (especially respiratory tract) and cancer. Smoking and nicotine may induce hepatic drug metabolizing enzymes that have been associated with increased antipyrine disappearance indicative of increased drug metabolism; thus, greater doses of drugs may be required to produce a therapeutic effect in smokers.

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Source: https://www.industrydocuments.ucsf.edu/docs/pzdl0000